Effect of Thalamocortical Activation on Recruiting Responses

I. Reticular Stimulation

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BERGER WAS the first to observe blocking of the spontaneous synchronized alpha waves in the human EEG following sensory stimulation, attention, or mental effort. He interpreted this as an inhibitory effect and maintained that the blocking was characteristic of all cortical regions not specifically excited by the sensory stimulation or involved in attention. Adrian and Matthews [1] confirmed the blocking of alpha waves by sensory stimulation but initially thought it was the result of desynchronization and temporal dispersion of cortical neuron firing. Bishop and Clare [8] interpreted spontaneous wave activity as "after-discharges" following repeated depressions associated with sensory volleys in cortical networks. More recently it has been postulated that the neural processes underlying alpha waves, spindle bursts and other synchronized waves, including recruiting and augmenting waves, might be dendritic and/or synaptic membrane potentials.

Much more is known about the factors which affect the state of synchronization of such waves than the basic mechanisms underlying them. Jasper [32] introduced the term *cortical activation state* to describe the slow potential shift associated with alpha blocking and Rheinberger and Jasper [64] used the term *activation pattern* to describe the low amplitude fast activity and absence of alpha waves in the cat EEG associated with the presentation of novel stimuli. Moruzzi and Magoun [49] demonstrated that sensory stimuli or direct electrical stimulation of the mesencephalic reticular formation by high frequency shocks cause activation or desynchronization of EEG background activity, either autonomous rhythms or sleep-like spindle bursts associated with certain stages of bar-

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biturate narcosis. This effect may operate via nonspecific thalamic nuclei [33, 68] and through extrathalamic pathways.[39, 67] Púrpura [54] pointed out that cortical arousal consists in an inhibition of cortical synapses, since the surface negative responses due to the activation of apical dendrites of cortical pyramidal cells [15] are blocked by arousal induced by the reticular formation. This notion implies two things: that reticular activation has its primary effect on cortical synaptic organizations, and that this effect is opposite to a surface depolarization, thus being hyperpolarizing or inhibitory in nature.

The same blocking effect has been found with other spontaneous and induced cortical "dendritic" potentials during reticular activation. The blocking effect has been shown on barbiturate spindles,[49] on responses induced by topical cortical electrical stimulation,[40, 46, 54] on strychnine cortical spiking,[49, 76] on primary and secondary evoked potentials,[12, 13, 22, 23, 27, 33, 34, 43, 49] on augmenting responses,[27, 52] and on recruiting responses.[26, 36, 37, 49, 50, 65, 74, 76] A careful review of these investigations shows that only the spontaneous waves and the spindles characteristic of sleep are abolished and replaced by low-voltage, fast activity. Evoked cortical responses are attenuated but not abolished. The effect of activation or arousal is on certain elements which appear to be modulated periodically during relaxed waking and sleep conditions.

The present investigations attempt to analyze the effect of activation on thalamocortical systems by means of the blocking of recruiting responses. Experiments were carried out according to the following program: (1) experiments characterizing the effect of reticular activation on cortical recruiting responses; (2) experiments comparing reticular activation with that induced by central and peripheral influences; (3) experiments determining the crucial role of the mesenphalic reticular formation on cortical activation.

The first series of experiments show that reticular activation consists in the blocking of the periodic, *waxing and waning*, modulation of the cortical responses induced by repetitive thalamic stimulation. This is a generalized reaction affecting all cortical recruiting areas. The second series of experiments show that activation appears to be the same whether induced by central neural stimulation (telencephalic, rhinencephalic, diencephalic, mesencephalic and rhombencephalic structures) or by peripheral sensory stimulation (visual, auditory, olfactory, nociceptive and proprioceptive). Finally, the third series of experiments show that lesions in the mesencephalic reticular formation enhance the waxing and waning aspect of the thalamocortical potentials and block thalamocortical activation regardless of the way in which it is induced.

These results suggest that activation and arousal are a unitary phenomenon probably limited to certain synaptic events in the cortex and mediated by mesencephalic reticular,[49] thalamic [33] and thalamo-orbitofrontal [69-72] systems.

Method

The first group of experiments was performed on 30 cats, protected by local anesthesia, and immobilized with Flaxedil (gallamine triethiodide). These experiments consisted in the induction of cortical recruiting responses and the observation of the effect on recruiting of high frequency stimulation of the mesencephalic reticular formation.

Initially, under deep ether anesthesia, tracheal and saphenous vein cannulas were introduced, and a routine craniotomy was performed. After completion of surgical procedures and the infiltration of wound margins and pressure points with a 0.2 % solution of procaine hydrochloride, ether was discontinued. The experiments began two hours after cessation of ether anesthesia.

Recruiting responses were produced by stimulation of the diffuse thalamic projection nuclei and were recorded at the surface of the cortex. Thalamic stimulating electrodes were parallel stainless steel wires insulated with teflon except at the tips which were separated by 0.25 mm, the diameter of the non-insulated tip being about 100 microns. Eight different thalamic nuclei were stereotaxically explored by stimulation of 45 different sites, ranging from Fr 7.0 to Fr 13.0, Lat. 0.0 to Lat. 4.5, and H +0.5 to H +3.0 (Horsley-Clarke coordinates). These nuclei included centrum medianum (CM), N. centralis medialis (NCM), N. centralis lateralis (CL), N. reuniens, N. paracentralis and N. rhomboidalis, ventralis anterior (VA), and N. reticularis (R). Thalamic stimulation consisted in trains of monopolar square wave pulses of 0.5 to 1.0 msec duration at a frequency of 8-10/sec. The intensity was adjusted in order to elicit bilateral cortical responses.

In each experiment, cortical responses were recorded from bilateral and symmetrical cortical areas, including anterior sigmoid (AS), posterior sigmoid (PS), anterior suprasylvian (ASS), posterior suprasylvian (PSS), and sometimes the lateral (L) and ectosylvian (ECTO) gyri. All cortical responses were recorded monopolarly with the reference lead attached to the stereotaxic frame. In twenty experiments, cortical electrodes consisted of blunt-end stainless steel screws inserted in the skull and in contact with the dura. In ten experiments, silver-silver chlorided ball-tipped electrodes, touching the pial surface of the exposed cortex, were used.

In addition to recruiting responses, in six experiments, augmenting responses were produced by stimulation of N. ventralis lateralis (VL) and were recorded at the anterior sigmoid gyrus. In all experiments, arousal was produced by stimulation of the mesencephalic reticular formation (FR 2, Lat. 1.0 to 2.0, and H —1.0 to —4.0). Reticular stimulation was always ipsilateral to the thalamic stimulation. Stimuli consisted in 100 to 150/sec trains of monopolar square pulses of 0.5 to 1.0 msec duration. The intensity was never more than 3 volts. Thalamic and reticular stimulation was administered by Grass S4 stimulators through Grass SIU4 isolation units. Cortical responses were simultaneously recorded on a Grass Model IIID eight-channel electroencephalograph and a Tektronix Model 502 dual beam oscilloscope with Model 122 Tektronix preamplifiers.

At the conclusion of each experiment, electrode placements were marked by passing anodal current (2 mA for 15 sec). The brain was then perfused with saline, followed by 10 % formalin. Thalamic and reticular placements were assessed by photographs of frozen sections by the Guzmán-Flores *et al* [29] technique and in Nissl preparations. Nomenclature and abbreviations describing placements are according to Jasper and Ajmone-Marsan.[35]

Results

Electrocortical arousal induced by reticular formation stimulation was observed by evaluating its blocking effect on cortical recruiting responses.

Blocking of recruiting was observed in the following way: Trains of thalamic 8-10/sec stimulation were applied to evoked recruiting responses lasting 3 to 5 seconds and separated by twenty to forty seconds. During the second train, reticular stimulation was applied, and the effect on recruiting was compared with the preceding and following trains. No blocking test trials were begun until recruiting returned to control values. Reticular stimulation was either initiated before and continued into the recruiting responses or was initiated during recruiting. In both cases similar effects were observed.

The analysis of the blocking of recruiting responses included study of (a) cortical incremental responses elicited by repetitive thalamic stimulation, and (b) the effect of reticular formation activation on these incremental responses, specifically those components which are blocked and those which are not blocked by reticular stimulation.

Recruiting Responses

Incremental responses evoked by diffuse thalamic projection system (DTPS) low-frequency stimulation were considered as recruiting responses only if they fulfilled the following criteria: (a) monophasic negative or polyphasic in which the main component is negative with preceding and following positive components much smaller in amplitude; (b) latency of the initial deflection over 15 msec; (c) inconsistent negative response to single shock; (d) bilateral and widespread in cortical distribution.

The cortical distribution of recruiting responses induced from different thalamic areas has previously been studied,[25, 30, 31, 33, 37, 48, 67, 73] but not systemmatically and with respect to the following characteristics.

In the present experiments using small-tip electrodes recruiting responses were classified according to the site of thalamic stimulation from which the responses were elicited, their relative amplitudes in different cortical areas and the effectiveness of reticular stimulation in blocking the responses. Three groups of responses were differentiated, which will subsequently be described under the headings Group I, II and III. The areas from which recruiting responses on the surface of the cortex were recorded and the region of the thalamus which elicited responses of Groups I, II and III are shown diagrammatically in figure 1.

Group I.
Midline Recruiting Responses.

Stimulation sites for this group (Fig. 1, circles) include the following regions of the midline nuclear complex,[60] extending from the caudal part of CM (Fr 7.0) to the rostral pole of the thalamus (Fr 13.5) and including NcM, Re, and mesial portions of CM, Pc, VA, MD, and R. Figure 2, NCM[0], illustrates the nature and cortical locus of the recruiting responses elicited from the midline thalamic sites of stimulation. At both ipsilateral and contralateral anterior sigmoid gyri (RAS, LAS) maximal amplitudes and similar latencies of these responses were obtained. Re-

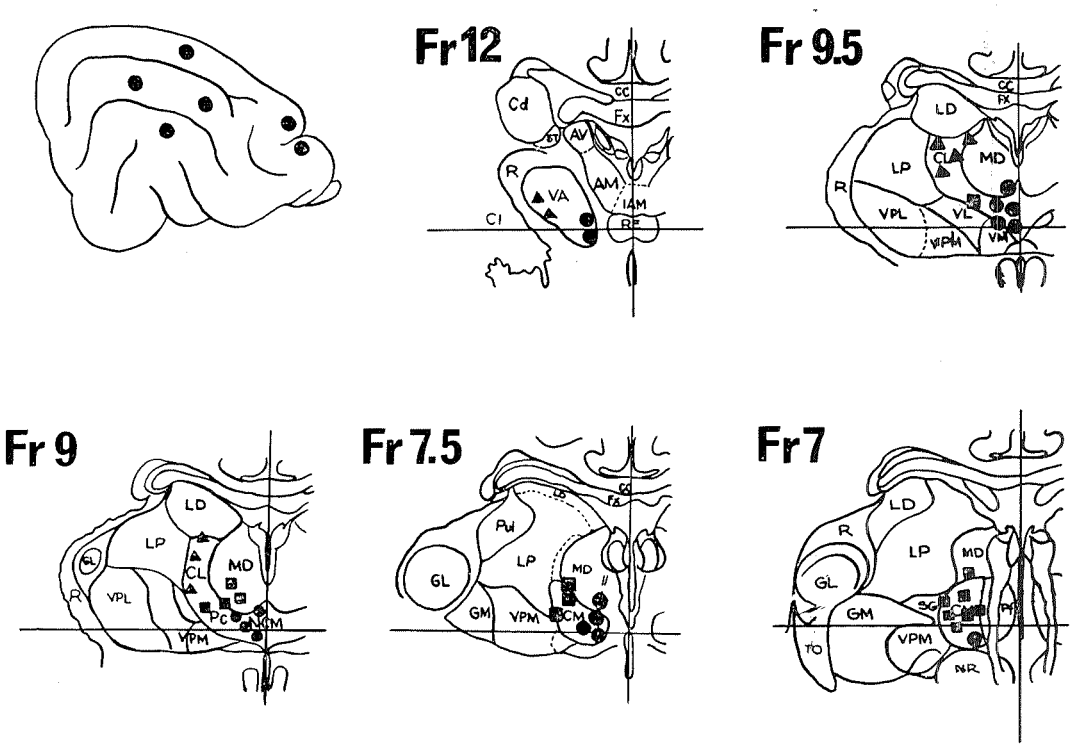
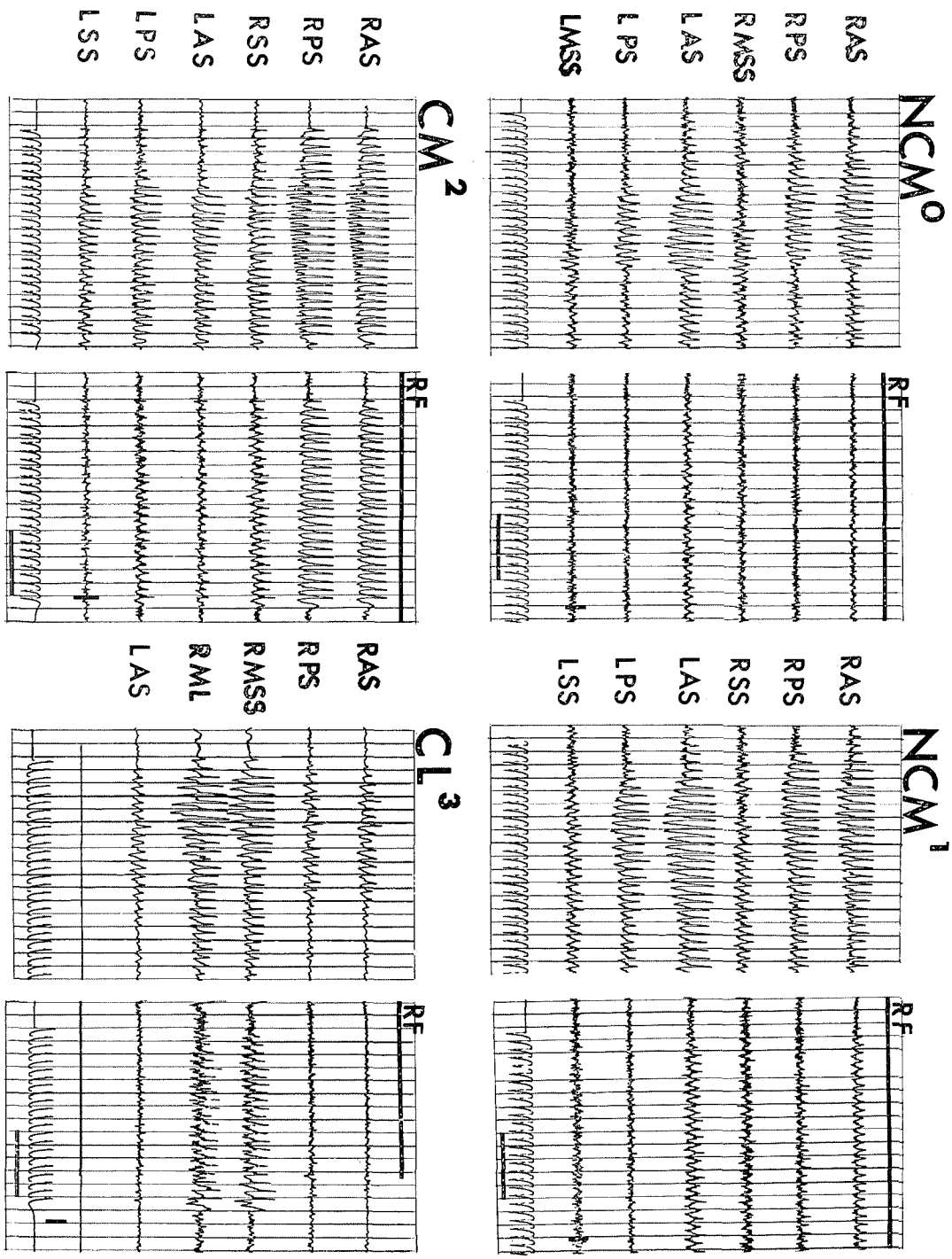


FIG. 1.— Diagram of dorsolateral surface of cat cortex showing recording sites for recruiting responses and diagrams from Fr 12 to Fr 7 showing sites of thalamic stimulation from which recruiting responses were elicited: Circles, group I recruiting responses. Squares, group II recruiting responses. Triangles, group III recruiting responses.

sponses in cortical areas posterior to the cruciate sulcus were always smaller in amplitude and not clearly delineated (RMSS and LMSS). The latency, development, and components of the responses varied with the intensity of thalamic stimulation, permitting classification of responses into two types.

Recruiting type I. Threshold recruiting responses had the following characteristics: monophasic, negative, latencies over 25 msec, and peak latencies from 45 to 55 msec (Fig. 2 and Fig. 3, NCM^0). The first shock applied to the thalamus failed to produce any cortical response. Subsequent 8/sec stimulation evoked cortical responses with slow development, requiring from 8 to 14 pulses to attain maximal amplitude. With continued repetitive stimulation, the responses decreased in amplitude until they almost disappeared and then again increased in amplitude, showing a slow periodic modulation (waxing and waning).

This type I recruiting, in the main, corresponds to the recruiting described in animals under medium or light barbiturate anesthesia [19-21, 33, 60] and in animals with mesencephalic reticular blockade.[14] It is of interest that type I


NCM 0
RAS
RPS
RMSS
LAS
LPS
LMSS
RF
CM 2
RAS
RPS
RSS
LAS
LPS
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RF
NCM 1
RAS
RPS
RSS
LAS
LPS
LSS
RF
CL 3
RAS
RPS
RMSS
RML
LAS
RF

FIG. 2.

recruiting is only elicited with certain frequencies of thalamic stimulation, i.e., 8-12 c/sec. Lower frequencies fail to evoke any cortical responses, and higher frequencies (15 or more per second) produce desynchronization.[33, 49]

Optimal conditions for recruiting type I responses included: complete relaxation and full local anesthesia for pain and pressure, warm environment, deprivation of sound and light, and slight hyperventilation. Under these favorable conditions, the EEG showed a synchronized 8-12 c/sec spindling activity and recruiting responses were easily produced at low intensities of thalamic stimulation.

Recruiting type II. These responses were elicited when the animal was slightly aroused or activated with a single shock to the reticular formation, thus producing desynchronization of the background activity. Under such conditions, threshold intensities of thalamic stimulation capable of inducing type I recruiting responses failed to evoke any cortical response. Recruiting responses, however, could be induced by increasing the intensity of stimulation. The responses elicited by suprathreshold stimulation showed different characteristics which varied as a function of the stimulus intensity (Fig. 2 and Fig. 3, NCM^1). The potentials were triphasic, and were formed by an initial positive component, a main negative component, and a later positive component. The initial and late positive components increased in proportion to the increase in the main negative component. The latency of the initial deflection decreased to 15 to 20 msec, and the peak latency of the negative component ranged from 30 to 35 msec. A single shock applied to the thalamus evoked a monophasic negative response which was sometimes followed by a rhythmical afterdischarge. Repetitive shocks produced maximal amplitude recruiting in only three to six pulses; the stronger the stimulus (2 to 8 V) the more rapid the rise to maximal recruiting amplitude. After attaining maximal amplitude, the responses declined slightly and then maintained constant, non-waxing and waning, amplitude. Finally, recruiting type II responses were obtained with a wider range of stimulation frequencies (2 to 14) than type I.

FIG. 2.— Amplitude and distribution of recruiting of different thalamic regions and differential blocking by reticular activation. *Top row.* Group I cortical recruiting responses elicited from midline nuclei; NcM (NcM^0, threshold stimulus; NcM^1, suprathreshold stimulus). Maximal amplitude recruiting is observed at right and left anterior sigmoid gyri (RAS and LAS); minimal recruiting posteriorly at mid-suprasylvian (RMSS, LMSS). *Bottom row left.* Group II cortical recruiting responses elicited parasagitally from right CM (CM^2, threshold stimulus). Maximal amplitude recruiting at ipsilateral anterior and posterior sigmoid gyri (RAS and RPS), *Bottom row right.* Group III cortical recruiting responses elicited from right CL (CL^3, threshold stimulus); maximal amplitude recruiting at ipsilateral suprasylvian and lateral gyri (RMSS and RML), minimal in frontal areas (RAS, LAS, RPS). *Far right.* 150/sec stimulation of the mesencephalic reticular formation (RF) blocks incremental portion of all responses regardless of cortical distribution and thalamic origin; only non-incremental portion of response remains. Calibration: 100 μV and 1 sec.

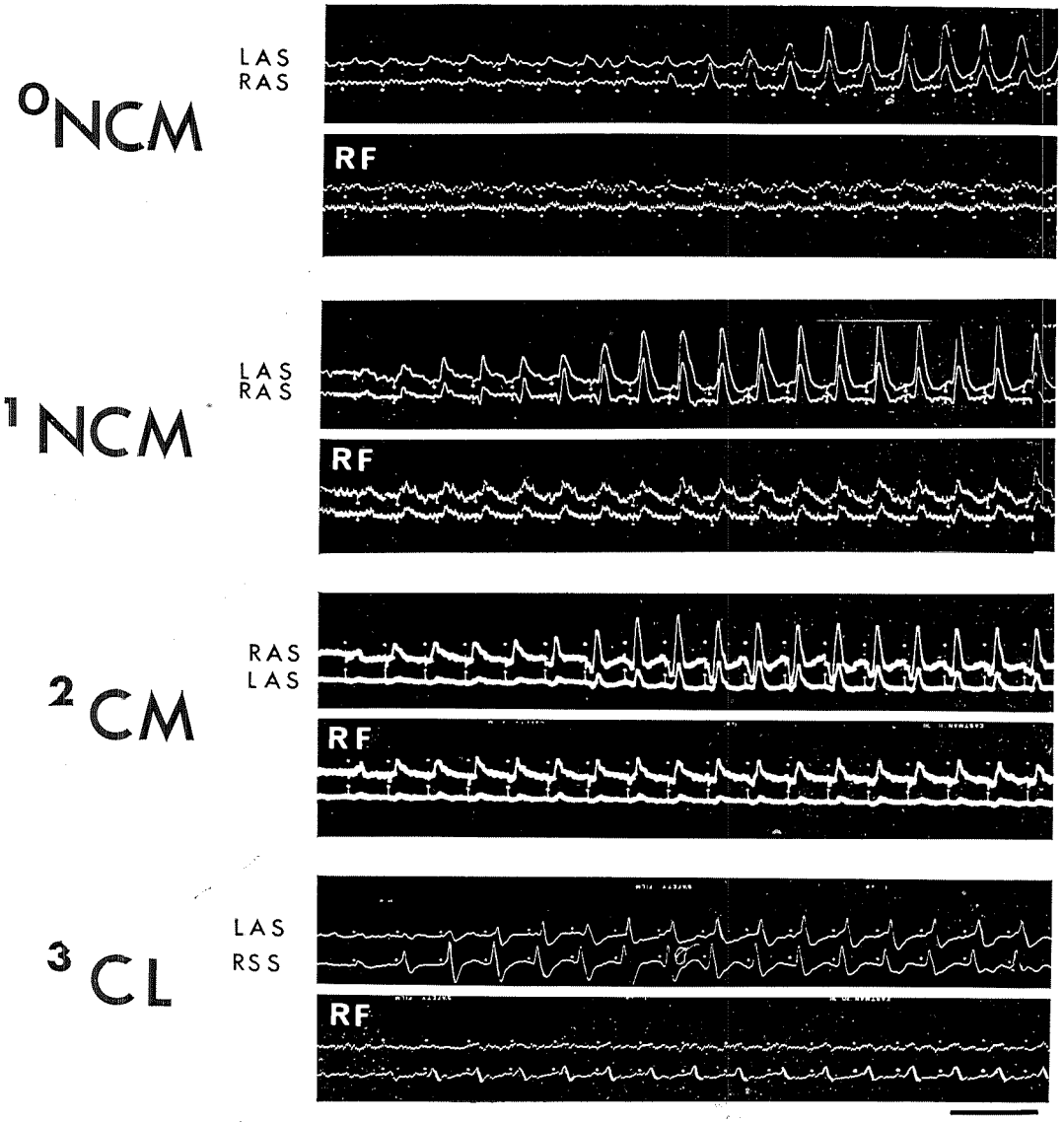

FIG. 3.— Nature of recruiting responses and differential blocking by reticular activation. *NcM⁰ and NcM¹*. Recruiting responses elicited at NcM and recorded in left (LAS) and right (RAS) anterior sygmoid gyri. Complete blocking of responses by reticular formation stimulation (RF) occurs on threshold recruiting (NcM⁰) and partial blocking occurs on suprathreshold recruiting (NcM¹). *CM²*. Recruiting responses elicited at right CM and recorded in right (RAS) and left (LAS) anterior sigmoid gyri. RF stimulation causes complete blocking of contralateral recruiting (LAS), partial blocking ipsilaterally (RAS) leaving only non-incremental portion of response. *CL³*. Recruiting responses elicited at right CL and recorded in left anterior sigmoid (LAS) and right suprasylvian (RSS) gyri. Complete blocking of response by RF stimulation occurs contralaterally (LAS) and partial blocking ipsilaterally (RSS). Calibration: 250 msec.

Group II.
Paramedial Recruiting Responses.

These responses were initiated by stimulation of paramedial DTPS nuclei, including lateral parts of CM, Pc, MD, and mesial parts of CL, between 2.5 to 3.0 mm lateral to the midline (Fig. 1, squares). Stimulation at these sites evoked recruiting responses which were maximal in the anterior and posterior sigmoid gyri, ipsilateral to the locus of thalamic stimulation (Fig. 2, CM^2). Contralateral responses were always smaller in amplitude and differed from the ipsilateral recruiting in several respects. The ipsilateral responses have characteristics similar to those of the type II recruiting responses (Fig. 3, CM^2). They showed a monophasic negative potential to the first shock. Repetitive 8/sec stimulation produced responses which quickly reached maximal amplitude in 3 to 6 pulses and which subsequently decreased slightly and maintained constant amplitude without periodic modulation. Responses were of short latency (15 to 20 msec) and showed initial and late positive components.

Contralateral responses, on the other hand, have characteristics similar to those of recruiting type I (Fig. 3, CM^2). They show either no response or only a very small response to single shock, a monophasic negative wave with latencies over 30 msec, and attain maximal amplitude in 8-12 pulses with a subsequent decline and waxing and waning amplitude.

Group III.
Lateral Recruiting Responses.

These responses were initiated in the lateral part of DTPS, specifically the lateral and dorsal part of CL and MD (Fig. 1, triangles). Maximal recruiting responses were obtained in midsuprasylvian and anterior ectosylvian gyri (Fig. 2, CL^3). Responses in these areas show features similar to those of type II recruiting responses (Fig. 3, CL^3).

In contrast, responses obtained in ipsilateral and contralateral frontal areas have features similar to type I recruiting responses.

Augmenting Responses

Augmenting responses are characterized by criteria established by others.[10, 14, 19-21, 30, 31, 60, 66] These responses may be elicited by stimulation of specific thalamic nuclei, and tend to be localized to a specific cortical area. A single shock produced a constant, biphasic, positive-negative potential of 1 to 2 msec latency (primary potential). Repetitive stimulation at 8 to 10/sec produced augmentation of the responses with an increase in amplitude and in peak latency. In general, these responses developed rapidly, attained maximal amplitude in 3 to 8 pulses after which the responses decreased (Fig. 4, VL).

Effect of Reticular Activation on Recruiting and Augmenting Responses.

High frequency stimulation of the mesencephalic reticular formation either abolished or reduced the amplitude of

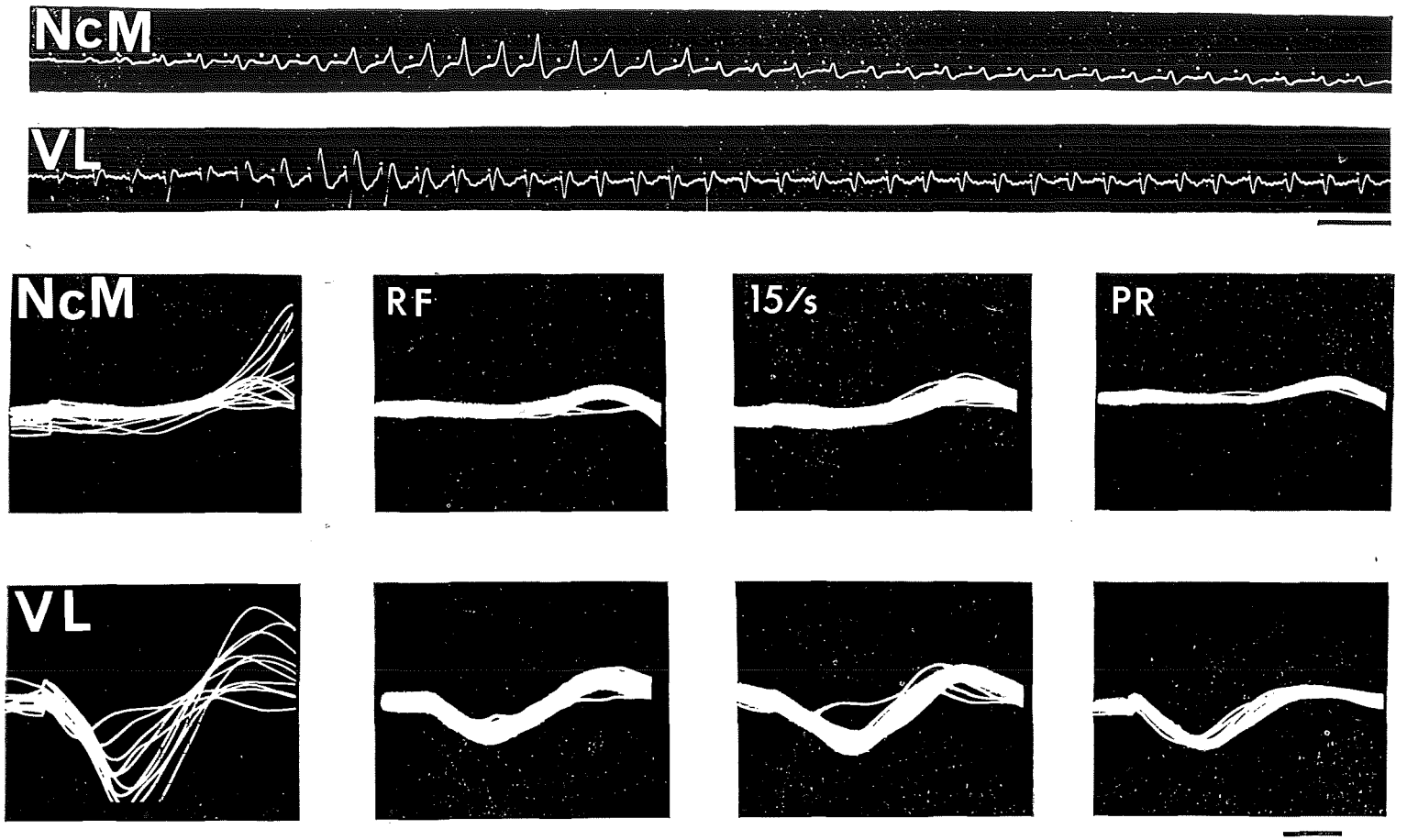

FIG. 4.— Blocked and non-blocked portions of recruiting and augmenting responses. *Top rows.* Cortical recruiting (NcM) and augmenting (VL) responses recorded from anterior sigmoid gyrus and induced by NcM and VL stimulation. *Bottom rows.* NcM recruiting and VL augmenting cortical responses elicited under the following conditions: (NcM) 8/sec stimulation; (RF) during reticular formation stimulation; (15/s) during repetitive 15/sec stimulation and (PR) during prolonged 8/sec stimulation. Reticular formation stimulation blocks only the incremental portions of both recruiting and augmenting responses. The remaining non-blocked portion is similar to the response observed when non-incrementation occurs, namely, during 15/sec thalamic stimulation or prolonged thalamic 8/sec stimulation. Calibration: top, 250 msec; bottom, 5 msec.

the recruiting responses. Stimulation at a single site in the reticular formation blocked all incremental potentials regardless of the thalamic origin of the responses or the cortical area from which they were recorded (Figs. 2, 3). The effect of RF stimulation was mainly upon the incremental portion.

Reticular formation activation abolished type I recruiting responses elicited by threshold stimulation of the midline thalamus. Complete blocking was also observed on recruiting responses contralateral to the locus of thalamic stimulation in the case of paramedial and lateral recruiting responses (Fig. 3, CM^2, CL^3).

Complete blocking of the recruiting responses was best exhibited when they were elicited by low intensities of thalamic stimulation. The blocking effect persisted several minutes after reticular stimulation; during this period recruiting responses could be restored only by increasing the stimulus strength.

Reticular activation capable of producing EEG desynchronization and complete blocking of type I recruiting did not abolish type II recruiting responses but only decreased the amplitude (Fig. 2 and Fig. 3, NCM^1). This same decremental effect was observed on ipsilateral recruiting responses induced by paramedial DTPS stimulation (Fig. 2 and Fig. 3, CM^2 and CL^3) and on augmenting responses induced by specific thalamic stimulation (Fig. 4, VL/RF).

In all cases where RF stimulation blocked recruiting or augmenting responses it was the incrementing portion of the response that was blocked or reduced. The remaining, non-blocked, portion of the response, which appears to be non-incremental to repetitive stimulation, has been observed under three conditions illustrated in figure 4, NcM; during stimulation of reticular formation (RF), during higher than normal frequency (15/s) of stimulation for recruiting, and during prolonged (PR) recruiting stimulation. In each of these conditions only a consistently low amplitude response persisted. The same effect of RF stimulation upon augmenting responses is shown in figure 4, VL.

It appeared, therefore, that repetitive thalamic stimulation produced two different processes at the cortical level: (1) direct or evoked cortical responses, and (2) a process of incrementation and modulation of the cortical responses. This latter process seemed to be evoked only at certain frequencies of stimulation [21] and only during the initial steps of development in awake preparations.[33] The attenuation of thalamocortical recruiting and augmenting responses during reticular activation consisted in a blocking of the incrementation process. Reticular stimulation did not abolish the non-incremental aspect of the responses even with voltages four to eight times greater than those used to block the incrementation.

Discussion

In the present experiments, the effect of reticular-induced activation on thalamocortical mechanisms has been

evaluated by studying the blocking of recruiting responses.[49] Blocking of recruiting was chosen as a means of evaluating electrocortical activation since it occurs during behavioral arousal, attention, and differential conditioning.[26, 75, 77, 78] Furthermore, blocking of recruiting has methodological advantages. Specifically, recruiting responses may be induced when desired and under controlled parameters of stimulation. The responses may be characterized in terms of latency, amplitude, components, and temporospatial development. Also the neural mechanisms underlying recruiting responses, although still in process of clarification, are better known than those underlying spontaneous potentials.[55, 56, 59] Thus blocking of recruiting may be observed when desired and may be classified and quantified accurately according to the blocked and non-blocked elements. The implications derived from a study of blocking of recruiting may be compared with the extensive literature available on the subject of thalamocortical recruiting.

The nature of reticular activation. Reticular activation mainly consists in the blocking of the incrementation and periodic modulation of the thalamically induced cortical responses. This effect is common to all observations and appears generalized regardless of the thalamic origin or the cortical distribution of the recruiting responses. Blocking of recruiting occurs differentially, however, according to the characteristics of the recruiting responses themselves: intensity and site of thalamic stimulation, cortical area where recruiting is observed, latency of recruiting responses, components of the responses, development of the incrementation, and periodic modulation.

Reticular activation abolishes only recruiting type I, induced by threshold midline thalamic stimulation, and contralateral recruiting, induced by parasaggital or lateral DTPS stimulation. It is important to note that none of these responses have concomitant discharges of the pyramidal cells.[60] On the other hand, reticular activation only blocks the waxing and waning features of any other type of recruiting or augmenting responses, which all have concomitant pyramidal and non-pyramidal cell discharges.[3, 4, 11, 14, 24, 38, 42, 45, 47, 51, 53, 60-62, 66]

The non-blocking element. A non-incremental response elicited by thalamic stimulation persists during reticular activation, even when the reticular formation is stimulated four to eight times more intensely than necessary to block the incremental aspect of the response. This non-incremental response appears monophasic or biphasic with a constant latency of the negative peak (32-35 msec). Under similar conditions, reticular activation fails to abolish augmenting responses. A positive-negative, short-latency, non-incremental response similar to the primary evoked potential persists during reticular activation. These results are in complete agreement with the observations of Gauthier *et al* [27] and Parma and Zanchetti [52] on the blocking of augmenting. According to Parma

and Zanchetti, the persisting element is accompanied by pyramidal discharge. This discharge occurs without incrementation during repetitive thalamic stimulation. In the present study, the non-incremental elements of both recruiting and augmenting responses which are refractory to reticular activation may be identified with the potentials evoked by repetitive thalamic stimulation under conditions in which no incrementation is observed, i.e., prolonged 8/sec stimulation and 15/sec stimulation to the thalamus. It has been assumed that the refractory elements represent a different phenomenon unrelated to the process of incrementation and probably due to activation of cortical synapses determining pyramidal discharges.

The blocking element. Reticular activation blocks the incremental and waxing and waning features of recruiting responses. Recent reports [69, 70-72] indicate that the process of incrementation and periodic modulation of cortical recruiting depends on a thalamo-orbital system regardless of the passive and active nature of its cortical events. [2, 6, 8-10, 14, 16-18, 28, 44, 55, 56, 58-62, 63, 66] We therefore might postulate that repetitive thalamic stimulation sets up two different processes. (1) It drives non-incremental cortical responses, accompanied by activation of pyramidal cells, which are not blocked by reticular activation. (2) It induces a process of incrementation and periodic modulation of these responses, which is blocked by reticular activation. These processes seem not to be determined by discharges but are only modified by the excitability state of the cortex. Such processes have been interpreted as regulatory,[15, 41] timing,[44] or conditional [63] processes. Reticular arousal thus might be considered as the blocking of the periodic modulation of cortical excitability existing during drowsiness and sleep. This modulation probably occurs at the level of the thalamus and may be explained as a blocking by lateral inhibition processes [5] or as a temporal dispersion of fixed IPSP patterns of thalamic neurons.[57]

Summary

In 30 cats immobilized with gallamine, the effects of reticular activation on thalamocortical systems has been evaluated by an analysis of blocking of recruiting responses. Recruiting responses were elicited by repetitive 8/sec stimulation of the intralaminar thalamic nuclei and were recorded in various cortical areas. Responses were classified according to the cortical distribution, thalamic origin, and susceptibility to blocking by reticular stimulation. Individual recruiting characteristics were analyzed in regard to latency, components, and temporo-spatial development (incrementation and periodic modulation). Cortical activation was induced by high frequency stimulation of the mesencephalic reticular formation. The results and conclusions may be summarized as follows:

Recruiting responses showed different cortical distributions with respect to amplitude depending upon the site of thalamic stimulation. In contrast reticular activation whether initiated before or during recruiting caused a generalized blocking effect on recruiting regardless of the thalamic locus of initiation or the cortical region of recording.

The effect of reticular activation was, however, limited to the incrementing portions of the cortical recruiting and augmenting responses. The remaining non-incremental portion of the response was characteristically observed during thalamic stimulation in which there was no incrementation, i.e., during prolonged 8/sec stimulation or during 15/sec stimulation just above the usual recruiting frequency range.

Repetitive thalamic stimulation appeared to induce two simultaneous but quite different cortical responses. One consisted in a directed or evoked cortical activity and the other consisted in an incrementation and periodic modulation of this activity.

It is concluded that reticular activation causes blocking of the incrementation process and interferes with periodic modulation of the amplitude of the cortical responses. Further it is postulated that reticular activation interferes mainly with a thalamocortical system which generates modulated waxing and waning potentials. This function may be inhibitory but also it may be explained in terms of a temporal dispersion of the elements participating in these evoked potentials. Finally, it is proposed that the non-blocked elements of the responses represent the activity of different synaptic events accompanying corticofugal discharges.

Resumen

Se evalúa en 30 gatos inmovilizados con gelamina, el efecto de la activación del sistema talamocortical, mediante el análisis del bloqueo de la respuesta de reclutamiento. Las respuestas de reclutamiento fueron inducidas por una estimulación repetitiva a 8/sec de los núcleos intralaminares y registradas en varias áreas corticales. Se clasificaron las respuestas con respecto a su distribución cortical, origen talámico y susceptibilidad a ser bloqueadas por la estimulación reticular. Los reclutamientos individuales fueron analizados con respecto a latencia, componentes y desarrollo temporoespacial (incremento y modulación periódica). La activación cortical se produjo por estimulación de alta frecuencia de la formación reticulada mesencefálica. Los resultados y conclusiones pueden ser resumidos como sigue:

La amplitud de la respuesta de reclutamiento mostró uno diferente distribución cortical de acuerdo al lugar de estimulación talámica. En cambio la activación reticular iniciada antes o durante la respuesta de reclutamiento produjo un efecto bloqueador generalizado del reclutamiento sin importar el locus talámico de iniciación o la región de registro cortical.

El efecto de la activación reticular se limitaba a los sectores de incremento de las respuestas de reclutamiento o aumento. La respuesta remanente, sin incremento, se observaba en las estimulaciones talámicas en que no se evidenciaba incremento, por ejemplo, durante la estimulación prolongada a 8 c/s o en estimulación a 15/s justamente por encima del rango de frecuencia que produce habitualmente reclutamiento.

La estimulación talámica repetitiva pareció producir dos respuestas simultáneas, pero diferentes. Una de ellas consistió en actividad cortical directa o evocada y la otra en un aumento o modulación periódica de esta actividad.

Sen concluye que la activación reticular produce bloqueo del proceso de incremento e interfiere con la modulación periódica de la amplitud de la respuesta cortical. Además, se postula que la activación reticular interfiere principalmente con el sistema talamocortical que genera potenciales modulados con aumento y disminución periódica de amplitud. Esta función puede ser inhibitoria, pero puede ser también explicada en términos de una dispersión temporal de los elementos que participan en estos potenciales provocados.

Finalmente, se propone que los elementos de la respuesta que no se bloquean representan la actividad de elementos sinápticos que acompañan la descarga corticífuga.

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